CITY AND COUNTY OF DENVER

DEPARTMENT OF ENVIRONMENTAL HEALTH

ENVIRONMENTAL PROTECTION
1391 Speer Boulevard, Ste. 700
Denver, CO 80204
(303) 285-4053 FAX: (303) 285-5621

WELLINGTON E. WEBB
Mayor

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EPA REGION VIII
SUPERFUND BRANCH

September 7, 2000

Ms. Bonnie Lavelle
U.S. Environmental Protection Agency
Region VIII
999 18th Street, Suite 500
Denver, CO 80202-2466

RE: Draft Baseline Risk Assessment
Vasquez Boulevard/I-70 Site

Dear Ms. Lavelle:

Thank you for the opportunity to review the draft report titled Baseline Human Health Risk Assessment, Vasquez Boulevard and I-70 Superfund Site, Denver, CO, prepared by ISSI Consulting Group, Inc. We appreciated the opportunity to discuss and gain clarity on technical issues regarding the baseline risk assessment (BRA) with your contractor. We have the following comments:

General:

1. It is important that the contaminated properties of known and predicted health risk be cleaned up promptly to health-protective levels. The contamination found in the affected neighborhoods can have serious health consequences if left in place, and must be remedied. At the same time, we recognize that some additional data are necessary to understand site risks, and to ensure that cleanup dollars achieve effective risk reduction. We urge EPA to move forward with cleanup, while conducting public health responses and any necessary studies to assure that the remedy is protective in the short- and long-term.

2. The BRA provides a great deal of information, but remains a highly technical document that is somewhat difficult to understand. The BRA indirectly addresses several issues that have been discussed extensively in workgroup settings, such as exposure to pica children and arsenic toxicity for sub-acute and sub-chronic exposures. EPA needs to clearly state how it is addressing these issues, and the uncertainties surrounding these issues, so that community members and non-toxicologists can clearly understand EPA's approach, and any differences in approach from that recommended by other agencies. Providing effective community outreach and information is extremely important, so that all community members are informed and can understand site

issues. Community outreach and educational activities must be extensive and available to all neighborhood residents.

3. Numerous issues have been raised regarding whether cleanup levels established to protect human health in the long-term are sufficiently protective to address sub-acute and sub-chronic health risks. These issues contribute to uncertainty regarding cleanup levels needed to protect human health for shorter-term health risks. While the BRA uses an estimate of sub-acute and sub-chronic exposure that appears to be conservative, data regarding upper-bound estimates of sub-acute and sub-chronic exposure are not available, and are not likely to become available soon. We understand that EPA has convened a national workgroup, which ATSDR participates in, to address sub-acute and sub-chronic arsenic toxicity issues. However, apparently this workgroup has not made recommendations regarding appropriate toxicity factors. In the near future, it does not seem likely that there will be agreement between the various agencies regarding acceptable estimates of sub-acute and sub-chronic exposure, or estimates of sub-acute and sub-chronic arsenic toxicity.

   EPA must move forward in cleaning those properties determined to be of chronic health concern. Cleanup must not be delayed for those properties of chronic health concern while agencies attempt to reach resolution on sub-acute and sub-chronic exposure assumptions, or sub-acute and sub-chronic toxicity values. Typically, cleanup actions that address chronic health concerns are also protective of sub-chronic or sub-acute health concerns.

4. The screening approach taken in the BRA to estimate risks associated with sub-chronic or sub-acute exposures should be expanded and clarified. EPA should clarify whether a cleanup designed to address chronic health concerns is also protective of sub-chronic or sub-acute health concerns, and under what situations a cleanup that addresses chronic health concerns may not address sub-acute and sub-chronic concerns. This would add focus to future investigations and health studies. If the predominant concern is exposure rates, ATSDR must investigate the prevalence of pica children or others with high exposure levels. If sub-chronic or sub-acute toxicity factors are important, the national workgroup on sub-acute and sub-chronic arsenic toxicity issues must resolve those issues.

5. We understand that EPA and ATSDR are considering public health responses to better understand health issues and provide education regarding ~~arsenic health~~ issues to the affected ~~communities~~. We believe these responses must include: (a) health studies that address the potential health effects of the contaminants of concern, and also investigate whether exposure to these contaminants could be a factor in disease prevalence in the affected communities; (b) extensive health-related information and education to help community members understand how the site may affect or has affected their health, and ways to minimize exposure; and (c) medical monitoring or surveillance for community residents so they can understand their individual health status and whether they have been affected by the contamination.

6. Lead exposure is a significant public health concern in the affected neighborhoods. The relationships between exposures to lead sources and elevated blood lead levels in children are complex and can depend on a variety of factors. The best way to evaluate this relationship is with site-specific data. At this site, the lead risk assessment appears to be driven almost entirely by default assumptions in the IEUBK model. While the model is almost certainly conservative,

use of default assumptions is not likely to reveal the most effective way of achieving decreased blood lead levels in children. A great deal of data are available currently, and additional data are shortly to be available regarding site-specific blood lead levels and exposure factors. These data include medical monitoring and soil data from the adjacent Asarco Globe site, data from the 1996 Denver Childhood Blood Lead Survey (CDPHE 1996), blood lead data from the VB/I70 area currently available through the CDPHE Lead Poisoning Prevention Program (LPPP), and blood lead data soon to be collected by the CDPHE LPPP. We urge that the data be considered and evaluated before reaching firm conclusions about likely sources of lead exposure in the VB/I70 area. This will allow the EPA to focus on the most effective and protective ways to reduce blood lead levels in children, by addressing the source(s) of greatest concern. .

Specific:

7. Tables and figures should contain sufficient titles and descriptors so they "stand alone". For example, the tables interspersed throughout the text are often difficult to understand because they lack titles and complete descriptors. Please add units to all tables and figures where they have not been provided (e.g., Table 2-1, Table 2-4). Similarly, please define acronyms when they are used in a table or figure (*e.g.,* Table 4-3, "EPC = exposure point concentration").

8. *Section 2.1, Phase I/phase II grab sample investigation, Figure 2-1.* Please add the (average) detection limits for arsenic and lead as a footnote to the table, so that non-detections (NDs) are put into perspective.

9. *Section 2.3.1, Spatial patterns of contamination, Page 6.* EPA notes that the pattern of lead contamination at the intensively sampled properties followed a "boundary effect" similar to that of arsenic. We presume that EPA's source characterization study will address information on lead as well as arsenic, in an attempt to define source, extent, and potential for exposure.

10. *Section 2.3.2, Contaminant levels in other environmental media - Indoor dust, Page 6.* The discussion of interior dust data begs the question "...Is there a correlation between concentrations of arsenic or lead in attic dust and outdoor soil?". While not as important as interior dust from an exposure perspective, please clarify.

11. *Section 2.3.2, Contaminant levels in other environmental media - Tap water, Page 7.* Please add a discussion of available data for concentrations of lead measured in tap water in "high-risk" Denver homes. Those data, collected by Denver Water over the last eight years, indicate that even in high-risk metro-area homes, built between 1982-87 with lead-soldered copper pipes, or containing lead supply lines, lead levels are below 15 μg/L for first-flush water samples (approximately 850 samples in more than 100 homes) (Rose, M., 2000, personal comm.)

12. *Section 2.3.3, Biomonitoring*, Page 7. Please provide the ages of the individuals tested for the results presented in the table on the bottom of the page. This information would aid in interpreting the biomonitoring results, especially for lead in blood.

13. *Section 2.4.1, Concentration in sieved and unsieved fine soil samples, Page 8.* The text on page 8 describes bulk samples as being sieved to remove particles "larger than 10 mm", while bulk samples are labeled as "<2 mm" in Figure 2.4. Please clarify and correct as appropriate.

14. *Section 2.4.2, Speciation of arsenic and lead.* On page 9, the reference describing sample collection, preparation and analysis appears to be incorrectly listed as USEPA 1999c. Please correct.

15. *Section 2.6.2, Residential dust sampling.* Please provide a brief summary of the dust sampling methodology in the text, to aid in interpretation of the results. Also, because the interior dust samples were collected in October and November, please comment on potential biases in the data, if any, that may have been introduced by seasonality issues.

16. *Section 2.6.3. Residential garden sampling, Page 15.* Please provide a brief summary of the vegetable sample collection and preparation methodology, as a basis for a further discussion of associated uncertainties in Section 4.4.3.

17. *Section 2.6.3. Residential garden sampling.* In Figure 2-10, please present the correlation coefficients for the yard and garden soil data, so that readers can then judge the data correlation for themselves. Similarly, please add *p* values to the discussion on correlation of garden soils and vegetables, where statements are made as to statistical significance (page 15).

18. *Section 3.1. Conceptual Site Model (Figure ES-3, Figure 3.1).* The conceptual site model (CSM) designates a number of pathways as complete and potentially significant that require quantitative evaluation. However, many of those pathways are not evaluated in this BRA (*e.g.*, ingestion of surface water and sediment). Please clarify by distinguishing pathways that are to be addressed in this BRA, from (significant) pathways that are to be addressed in other risk assessments.

19. *Section 3.1. Conceptual Site Model (Figure ES-3, Figure 3.1).* Please correct the incomplete text in footnote "b" of the CSM.

20. *Section 3.2. Pathway Screening- Dermal contact with soil. Page 18.* Please verify and/or correct the citation provided in the text ("EPA 1995a") as the source for Region VIII's recommendation regarding assessment of dermal uptake for metals in soil.

21. *Section 3.2.2. Workplace exposures.* We agree that risk-based concentrations for workplace exposures may appropriately be set at values higher than those for residential exposures. However, because the source characterization has not yet been completed, we believe it is inappropriate to dismiss further evaluation of the workplace scenario, at this time. We recommend that EPA address the potential for workplace exposures in a future document, after the source characterization is complete, and data are sufficient for agreement on the likelihood of health concern from site-related chemicals, for this scenario.

22. *Section 4.2.4. Quantification of exposure of residents - Soil and dust ingestion, Page 25.* As stated above, tables in the text should contain titles. Please add a title to the table on top of the page (e.g., "recommended assumptions for ingestion of soil and dust").

23. *Section 4.2.4. Quantification of exposure of residents - Soil and dust ingestion, Page 26.* For clarity, please provide a summary table presenting values used in the soil/dust exposure

calculations at the end of the discussion on ingestion of soil and house dust (e.g., percent intake of soil vs. dust, fraction of indoor dust derived from outside soil, baseline (D0) dust value, etc.).

24. *Section 4.3.3. Adjustments for relative bioavailability.* A document provided to the working group in a recent technical meeting (Tsuji, J., 2000, personal comm.)described an error in the bioavailability calculations, as presented in the swine study (ISSI, 2000). Please review all swine study calculations, and make corrections to the bioavailability estimates, as appropriate.

25. *Section 4.3.3. Adjustments for relative bioavailability.* It is our understanding that bioavailability adjustments are made to account for differences in exposure, not toxicity. Therefore, we suggest the BRA text clarify that, while adjustments are made to toxicity factors (RfD_{adj}, SF_{adj}) for mathematical convenience, the experiments are designed to estimate residents' exposure to the particular form(s) of arsenic present in site soils.

26. *Section 4.4.2. Risks from soil and dust – Cancer Risk, Page 33.* While the working group was involved in the sampling design for soil, the BRA text should provide a brief summary of the justification for calculating an exposure point concentration from the 95% UCL of the mean for 3 composite samples.

27. *Section 4.4.2. Noncancer risks from short-term exposure, Pages 34-36.* For clarity, please add a table summarizing the assumptions used in the calculations for short-term arsenic exposures. Perhaps the information on short-term exposure (existing text and new table) would be most appropriately presented in the existing section on *Quantification of Exposure of Residents* (Section 4.2.4).

28. *Section 4.4.2. Exposure frequency, Page 35.* Please add text that clarifies the meaning of assuming "...1 day out of two", for the sub-acute exposure frequency. It is not intuitively obvious why exposure occurring on one day should be averaged over two, while it may be more obvious that exposure occurring over three days within six should be averaged over the intervening days. For example, exposure occurring on Monday, Wednesday, and Saturday should be averaged over the cumulative six days (3/6), while exposure occurring only Monday should not be averaged over Monday and Tuesday (1/2).

29. *Section 4.4.2. Toxicity factors, Pages 35-36.* From the brief information presented in the BRA, it is not apparent that appropriate toxicity factors were selected for sub-acute and sub-chronic exposure. All relevant toxicity data, including those presented in the recent scientific literature, should be considered in selecting appropriate arsenic toxicity factors for sub-acute and sub-chronic exposure. We suggest that EPA further document the values used in the BRA (after incorporation of an appropriate safety factor, as described in the following comment) and consider them as screening values, and revisit the issue when peer-reviewed toxicity values are issued by EPA's national arsenic workgroup.

30. *Section 4.4.2. Toxicity factors, Page 36.* ISSI has presented a LOAEL for sub-acute exposure in the text (i.e., 0.05 mg/kg-day) and adopted it for use as the sub-acute RfD, without the application of a safety factor. A safety factor should be applied when deriving a reference dose from a LOAEL, as described in USEPA guidance (e.g., USEPA 1993).

31. *Section 4.4.2. Results, Page 36.* The values in the results table in the middle of the page need better descriptors. The two far-right columns should be labeled as the "number of properties" for the values presented in the sub-acute and sub-chronic categories.

32. *Section 4.4.3. Risks from home-grown vegetables.* Please create a table (or add columns to an existing table) that presents arsenic intake from vegetable ingestion by property, along with the associated arsenic concentrations in garden soil and in yard soil.

33. *Section 4.4.3. Risks from home-grown vegetables, Page 36.* The text on the bottom of the page states that the 95% UCL of the mean arsenic concentration was calculated for "each property". Please add text clarifying what the sentence implies, i.e., that the UCLM was calculated by aggregating data across vegetable type.

34. *Section 4.4.3. Risks from home-grown vegetables, Page 37.* To better understand the uncertainty associated with the risk estimates for homegrown vegetables, please calculate arsenic intake from vegetables both with and without the values that appear to be outliers *(e.g.,* garlic at property 11).

35. *Section 4.4.3. Risks from home-grown vegetables, Page 37.* The following wording from the text is confusing. "An interview with the property owner did not reveal any probable source of arsenic in the garden." Does the author wish to state that the interview did not reveal any probable source for the arsenic, or that arsenic probably wasn't present at all?

36. *Section 4.4.3. Risks from home-grown vegetables.* Please provide a discussion of potential uncertainties associated with the vegetable sample collection and preparation methodology. In particular, address the lack of washing (beyond a field rinse with deionized water) and peeling, and the potential for vegetable results to be biased by adhering soil. Any future vegetable samples should be prepared for analysis in a manner that approximates typical consumer use (*e.g.*, peel onions and garlic, wash potatoes).

37. *Section 4.4.3. Risks from home-grown vegetables.* Please discuss the potential effect of estimating exposure by applying a single intake factor to all vegetable types, rather than an individual factor for each vegetable type. Please explain your rationale for that approach, and acknowledge the biases inherent in assuming equal intakes when concentration varies across vegetable type.

38. *Section 4.5. Uncertainties in arsenic risk assessment - Uncertainty in concentration terms. Page 38.* The document discusses uncertainties associated with estimation of concentration based on measurements of arsenic in bulk soil samples, rather than in the fine (sieved) fraction. However, there is no discussion of uncertainties associated with the use of the XRF technique, instead of a more traditional analytical approach, such as ICP. We suggest that the BRA include a discussion of uncertainties associated with the use of XRF data, and provide elaboration on potential cumulative effects that may result from biases associated with the combination of factors (e.g., use of XRF data, analysis of bulk samples).

39. *Section 5. - Exposure and Risk from Lead.* U.S. EPA guidance suggests incorporating appropriate site-specific data into the IE/UBK model when they are available (USEPA 1994,

1999). For example, if appropriate site-specific bioavailability data are available for lead in soil (e.g., from *in vitro* testing), they should be incorporated in the model. Also, EPA should use appropriate site-specific air and water data, if they are available, or discuss the limitations of the available data and explain why they are not appropriate for use. This is important, to gain an understanding of the relative contribution of each media to total lead exposure.

40. *Section 5. - Exposure and Risk from Lead.* As discussed in the technical meeting of 8-28-00, and agreed to by many of those participating, EPA should incorporate relevant data from CDPHE's Denver Lead Study (CDPHE 1996), medical monitoring data from Globe residents, as well as data collected from the VB/I70 site.

41. *Section 5. - Exposure and Risk from Lead. Table 5-1.* In the footnote, the baseline concentration of lead in dust is incorrectly stated to be 11 mg/kg. Please correct the footnote and verify that the appropriate baseline concentration (150 mg/kg) was used in the model calculations.

42. *Section 5.4, Uncertainties in lead risk evaluation.* The goal of evaluating lead exposures in the VB/I70 area is reduce blood lead levels in children by discerning the most important contributors to exposure, and implementing effective control strategies. It is important to use the most accurate site-specific data as inputs to the IE/UBK model, in order to understand contributions to exposure. As cited in the BRA, EPA's "three cities study" demonstrated that soil abatement alone was not effective for reduction of lead exposure, unless there "...is a substantial amount of lead in soil and unless this lead is the primary source of lead in house dust." If significant reductions in blood lead are to be achieved, the primary sources of lead exposure must be understood and then targeted for cleanup activities.

References:

CDPHE. 1996. Denver Childhood Blood Lead Survey. Final Report. Colorado Department of Public Health and Environment. January.

ISSI. 2000. Relative bioavailability of arsenic in soils from the VBI70 site. Public Review Draft. ISSI Consulting Group, Denver, CO. June.

Rose, M. 2000. Personal communication. Telephone conversation on 8-23-00 with G. Hook, Denver Department of Environmental Health, regarding lead testing results for Denver tap water. Denver Water, Denver, CO.

Tsuji, J. 2000. Personal communication. Email dated 8-9-00, to W. Brattin (ISSI) and C. Weis (USEPA) regarding errors in EPA's arsenic bioavailability study for VB/I70 site soils. Exponent, Bellevue, WA.

USEPA. 1993. Reference Dose (RfD): Description and use in health risk assessments. Background document 1A. Integrated Risk Information System. United States Environmental Protection Agency. March.

USEPA. 1994. Guidance manual for the Integrated Exposure Uptake Biokinetic Model for lead in children. United States Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC. February.

USEPA. 1999. Overview of the IEUBK model for lead in children. United States Environmental Protection Agency, Office of Emergency and Remedial Response. EPA 540-R-99-015. August.

Thank you for the opportunity to comment on this document. If you have any questions regarding these comments, please contact me at 303-285-4065, or Gene Hook at 303-285-4068.

Sincerely,

Celia VanDerLoop
Denver Department of Environmental Health

Cc:
Barbara O'Grady – CDPHE
Frances Hartogh – AGO
Bob Litle – Asarco
David Mellard – ATSDR
Laurel Mattrey– COPEEN
Chris Weis – EPA
Shaun Sullivan – CAO
Jane Mitchell - CDPHE
Anthony Thomas - Clayton
Michael Maes - Elyria
Chuck Patterson - Globeville
Sandy Douglas – Cole
Lorraine Granado – Swansea
Susan Muza – ATSDR
Cindy Bosco – EVS